COMPARATIVE STUDY OF EFFICACY OF MODIFIED VACUUM ASSISTED CLOSURE DRESSING USING GLOVES VERSUS BETADINE DRESSING IN CHRONIC NON HEALING ULCER IN GOVT RAJAJI HOSPITAL, MADURAI

Dissertation submitted to

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DEPARTMENT OF GENERAL SURGERY

MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL MADURAI–62502

CERTIFICATE

This is to certify that this dissertation entitled **COMPARATIVE STUDY OF EFFICACY OF MODIFIED VACUUM ASSISTED CLOSURE DRESSING USING GLOVES VERSUS BETADINE DRESSING IN CHRONIC NON HEALING ULCER IN GRH, Madurai"** at Government Rajaji Hospital, Madurai submitted by **DR SAKTHEESWARAN R** to the faculty of General Surgery, The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MS degree (Branch I) General Surgery ,is a bonafide research work carried out by him under my direct supervision and guidance.

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DR.S.R.DHAMOTHARAN M.S,FIAGES

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CERTIFICATE BY THE DEAN

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DECLARATION BY THE CANDIDATE

I declare that this dissertation entitled" **COMPARATIVE STUDY OF EFFICACY OF MODIFIED VACUUM ASSISTED CLOSURE USING GLOVES VERSUS BETADINE DRESSING IN CHRONIC NON HEALING ULCER IN GRH, MADURAI"** is prepared by me under the direct guidance and supervision of **Dr. A.M. SYED IBRAHIM M.S, FAIS,..,** Professor, Department of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI. This is submitted to **The Tamil Nadu DR.M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree(Branch I) General Surgery course on MAY 2019.

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DR.R.SAKTHEESWARAN

POST GRADUATE IN GENERAL SURGERY

INTRODUCTION

Negative pressure wound therapy or vacuum assisted closure dressing is a newer non-invasive technique that use controlled negative pressure, using vacuum-assisted closure (VAC) device. It helps to promote wound healing by removing fluid from open chronic wounds, preparing the wound bed for graft or other closure methods by reducing edema and promoting formation of granulation tissue. VAC dressing can be used to treat Chronic non healing ulcers following debridement of infection or amputation, and in reconstructive soft tissue and osseous procedures.

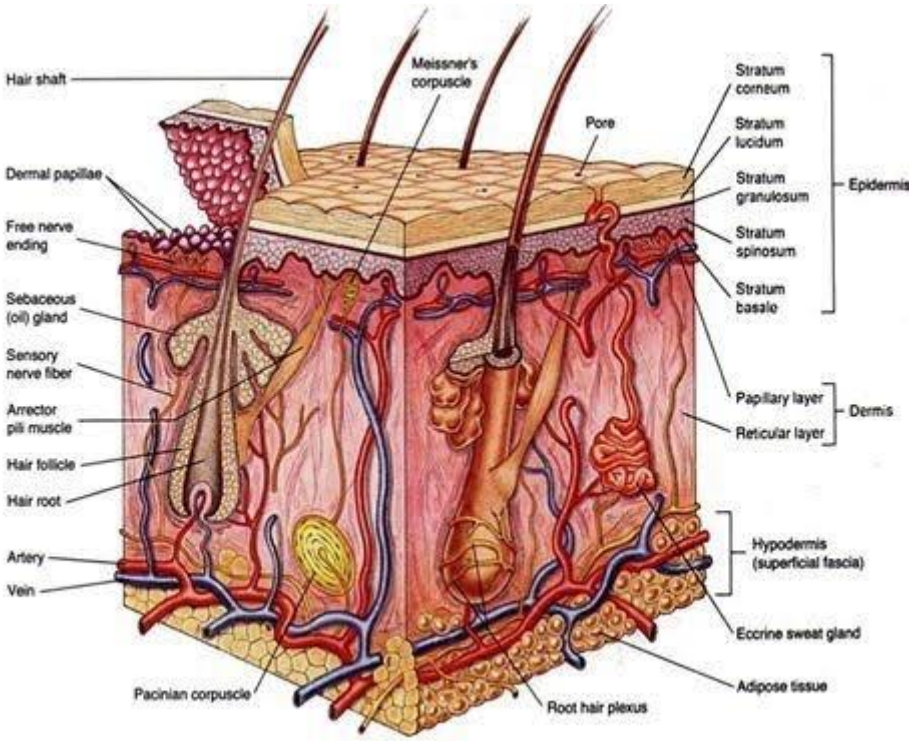
Vacuum assisted closure dressing has been frequently modified with given restrictions in resources available.

REVIEW OF LITERATURE

SKIN – ANATOMY

Skin is made of three layers

- Epidermis
- Dermis
- Subcutaneous tissue



Epidermis :

It is less than millimeter thick and made of three types of cells

Keratinocytes:

These cells from the basal layer towards the skin surface losses its water , begin to hard and dies eventually. The dead keratinocytes forms the outer most protective layer of the epidermis –stratum corneum which is sloughed off and replaced regularly.

Melanocytes :

Produces melanin pigment which is responsible for the colour of skin.

Langerhans cells:

Part of the immune system

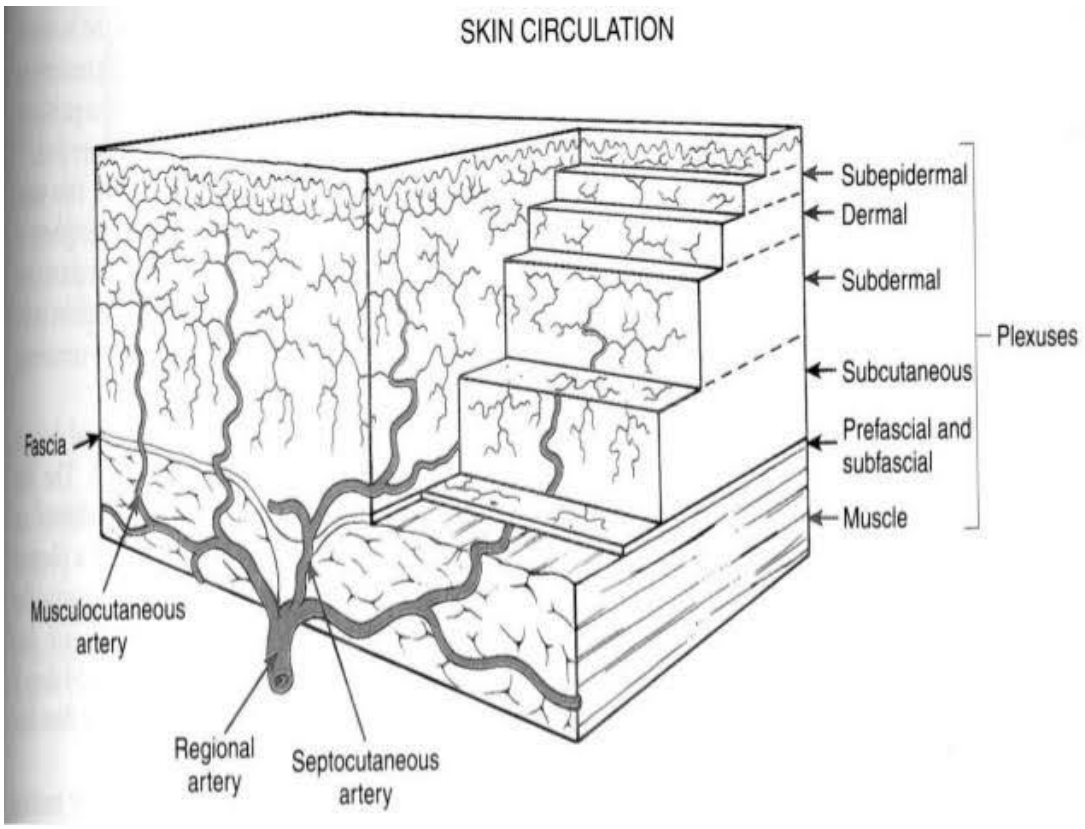
Acts as a defense against the pathogens entering the epidermis

DERMIS:-

It is the thickest layer of the skin. The primary cells present in this layer is fibroblast , which has collagen and elastin and gives skin its elasticity and resilience. It is the home of sebaceous gland and also contains capillaries and langerhans producing lymph nodes . Sebaceous glands produce sebum and it lubricates the skin surface.

SUBCUTANEOUS TISSUE:

It is composed of mainly adipose tissue responsible for the padding and insulation as well as contains sweat glands and erector pili muscle. Cutaneous vessels arising from named vessels supplies a 3-dimensional vascular territory from bone to skin called angiosomes. Cutaneous vessels anastomosis with nearby cutaneous vessels forming a vascular network within the skin



CHOKE VESSELS:

Choke vessels plays an important role in flap survival , as they provide initial resistance to blood flow between tip and base of the flap .

When the skin flap delaying done ,this choke vessels open up and provide adequate blood supply to the flap for the survival

FUNCTIONS OF SKIN

The skin has 4 major functions

1) PROTECTION – It provides protection agaist UV radiation , mechanical , chemical and thermal injuries. It acts as a physical barrier to microorganism invasion and prevents dehydration as it is relatively impermeable.
2) SENSATION – Skin is the largest sensory organ of the body and has the receptors for touch , pain , pressure and temperature.
3) THERMOREGULATION – Skin prevents heat loss by presence of hair and subcutaneous tissue . skin is the major thermoregulatory organ in human body . Heat loss is

facilitated by sweat evaporation from skin surface and increased blood flow through the rich vascular network in the dermis.

4) METABOLIC – Subcutaneous fat tissue contains major energy store mainly in the form of triglycerides. Vitamin D is synthesized in skin epidermis and also supplements from diet.

ANATOMY OF FOOT

A complete anatomical knowledge is essential while treating the non healing ulcers of the foot and its complications. Without aggressive management , non healing ulcer will end up in amputation either minor or major

The foot skeleton consists of tarsal-7 ,metatarsal – 5 and phalanges -14. Foot is usually divided into three zones

- FOREFOOT – metatarsal and phalanges
- MIDFOOT – navicular , cuboid and cuneiform
- HINDFOOT – talus and calcaneum

SKIN AND SUBCUTANEOUS TISSUES OF THE FOOT

The skin of dorsum of the foot is thinner and less sensitive while the plantar skin is thicker in weight-bearing areas such as heel , lateral margin and ball of foot and more sensitive and not pinchable . The sole of foot consist of thick stratum corneam and thin dermis. The subcutaneous tissue in the sole is more fibrous than dorsum of foot. Fibrous septa divides the sole into fat filled areas making the foot shock absorbable especially over the sole. The skin of the sole is hairless and has numerous sweat glands . the entire sole is more sensitive especially the thinner areas underlying the arch of foot.

The epidermis is usually transformed into the nail matrix. It contains three ill defined layers dorsal, intermediate and ventral layers. It is firmly attached to epithelium of nail bed .The margin of the nail is overhung by skin fold predisposing to in growing toe nails.

SKELETON AND FASCIA OF THE FOOT

The skeleton is shaped to form arches and adjust to uneven surfaces .It has 7 tarsal bones, 5 metatarsals and 14

phalanges. The superficial fascia of the sole is more fibrous and dense. Thick central part of the plantar fascia forms the plantar aponeurosis, resembles the palmar aponeurosis but tough dense and elongated.

The plantar aponeurosis fixes the skin of the sole and also helps in maintaining the longitudinal arches of the foot.

Compartments of the sole:

Medial compartment:- covered by medial plantar fascia which is thin and consist of abductor hallucis , flexor hallucis brevis , tendon of flexor hallucis longus , medial plantar nerve and vessels.

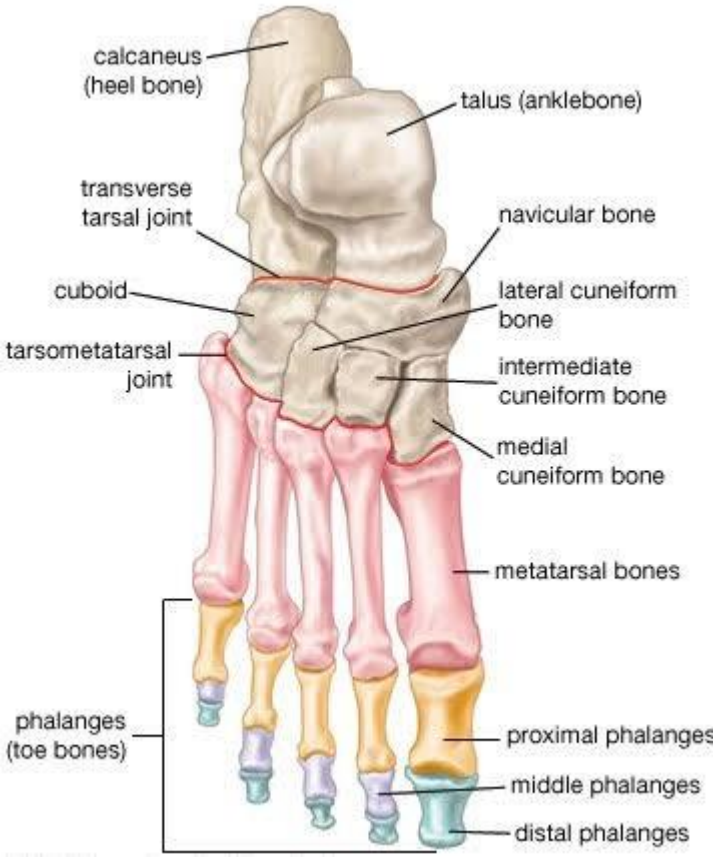
lateral compartment:- covered by lateral plantar fascia and consist of abductor digiti minimi and flexor digiti minimi

central compartment – covered by plantar aponeurosis and consisit of flexor digitorum brevis , tendons of flexor digitorum longus and flexor hallucis longus , lumbricals and adductor hallucis and also lateral plantar vessels and nerve

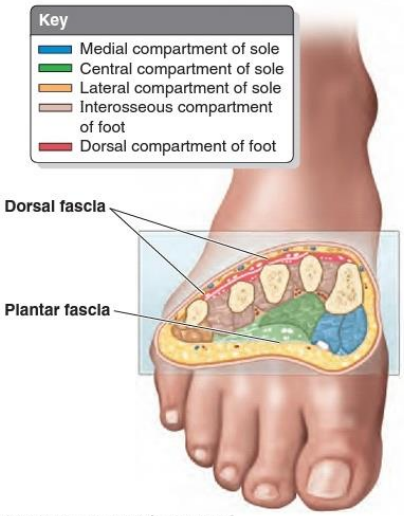
forefoot fourth compartment – interroseous compartment of foot : covered by dorsal and plantar interossei fascia and

consist of metatarsal bones , deep plantar and metatarsal vessels and plantar and dorsal interrosei muscles .

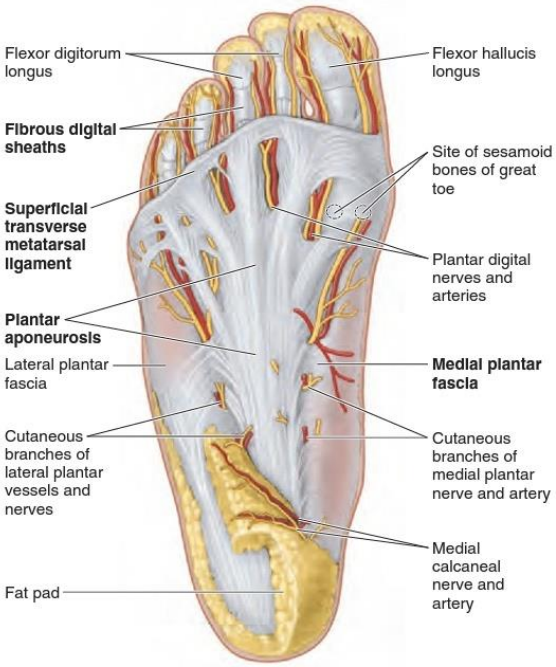
Fifth compartment or dorsal compartment:- it lies between dorsal fascia and tarsal bones and consists of extensor hallucis brevis and extensor digitorium brevis , neurovascular structures of the dorsum of foot.



(A) Fascia of dorsum of foot (anterolateral view)



(C) Transverse section of foot compartments (anterior view)



(B) Sole of foot (plantar aspect) (inferior view)

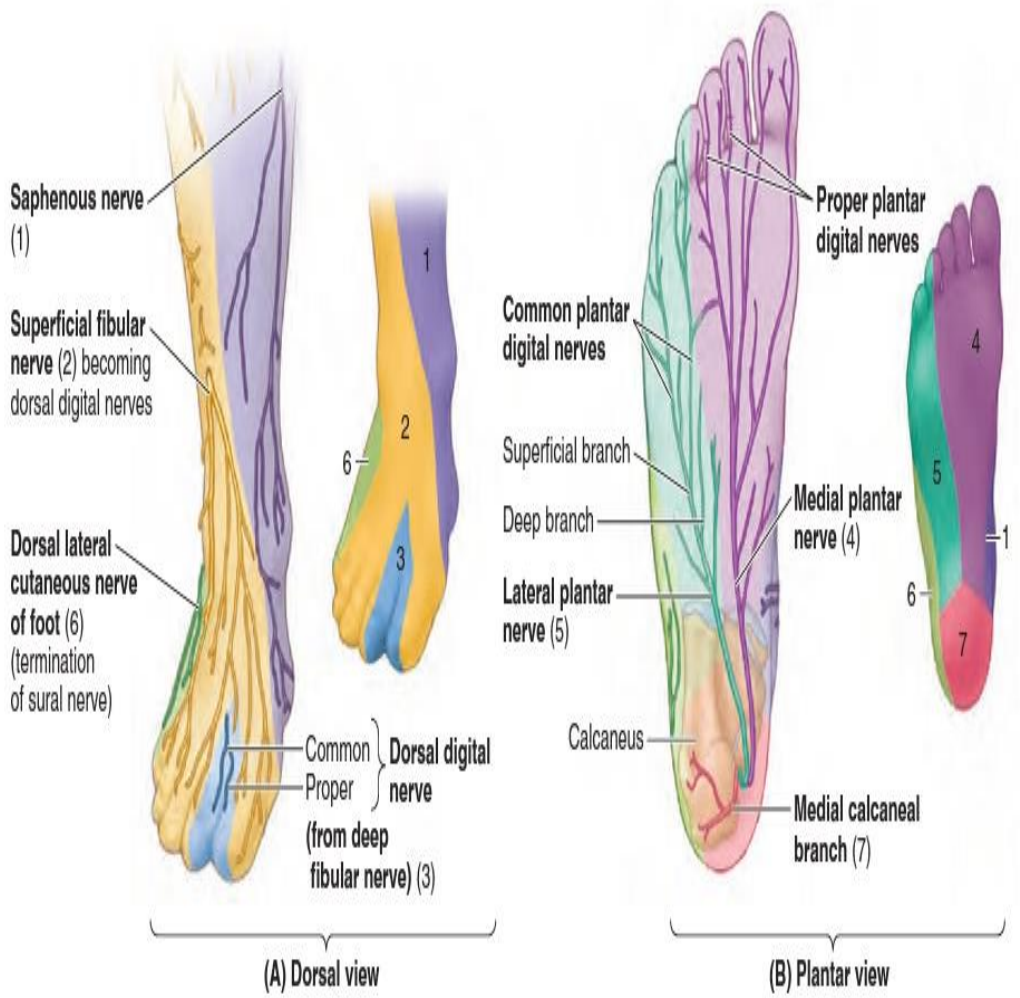
MUSCLES AND TENDONS OF THE FOOT

Despite , arranged in layers and compartment ,the muscles act as a group during the support phase of the stance and in maintaining longitudinal and transverse arches of the foot. These muscles become more active during the later phase of walking by stabilizing the foot for propulsive movement.

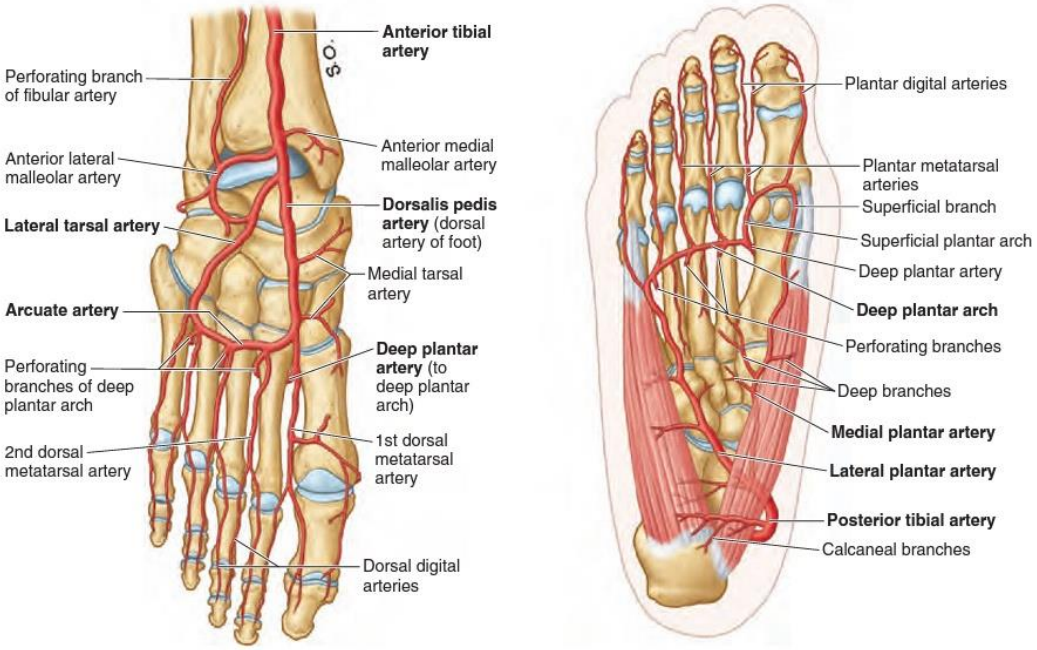
MUSCLES OF DIFFERENT COMPARTMENTS

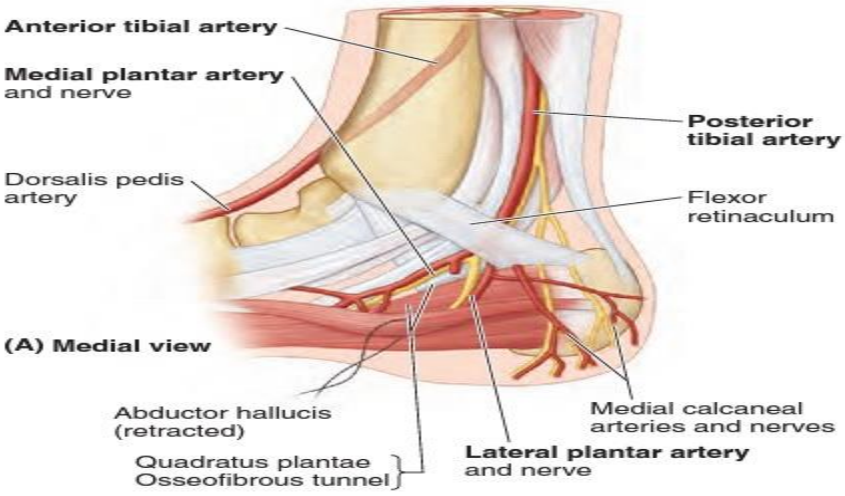
COMPARTMENTS	MUSCLES
1ST layer	Abductor hallucis , flexor digitorum brevis,abductor digiti minimi
2nd layer	Quadrates plantae, Lumbricals
3rd layer	Flexor hallucis brevis , adductor hallucis, flexor digiti minimi brevis
4th layer	Plantar interossei and dorsal interossei

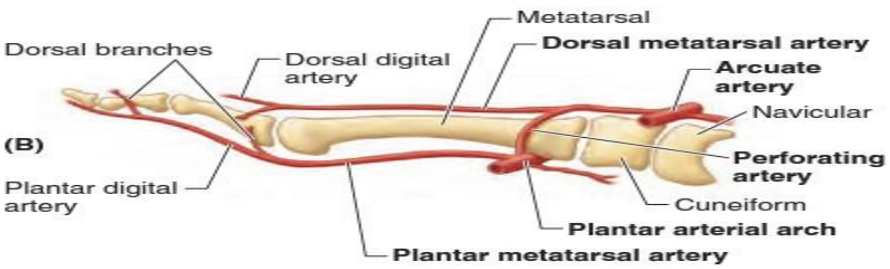
NERVES OF THE FOOT



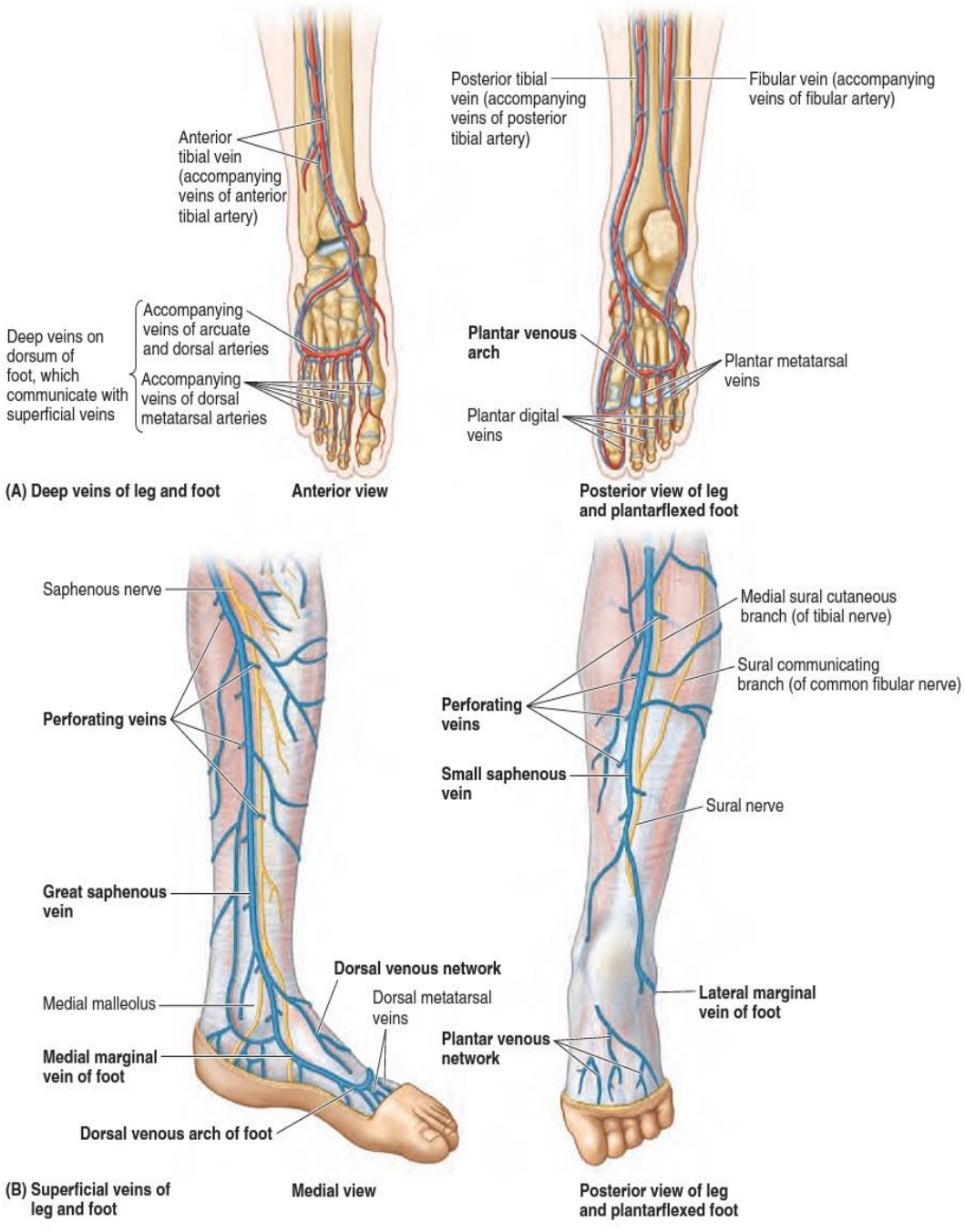
ARTERIAL TREE







VENOUS DRAINAGE



LYMPHATIC DRAINAGE

Superficial lymphatics are numerous in the sole of foot medial superficial lymphatics are large and accompany great saphenous vein to vertical group of inguinal nodes. Lateral superficial lymphatics accompany short saphenous vein and drains into popliteal nodes.

Deep lymphatics follows main blood vessels ; fibular anterior tibial, posterior tibial , popliteal and femoral veins into deep inguinal lymph nodes

ARCHES OF FOOT

These arches used to distribute the weight over the foot and acts as shock absorbers and spring boards for propelling while walking , running and jumping etc,,

Longitudinal arch – medial and lateral parts

Medial – is higher and formed by calcaneus , talus , navicular , three cuneiform and three metatarsals. Talar head is the keystone in maintaining medial longitudinal arch.

lateral – flat than medial arch .and formed by calcaneus , cuboid , and lateral two metatarsals

transverse arch formed by cuboid , cuneiform ,and **bases** of metatarsals

PASSIVE FACTORS FORMING ARCHES OF FOOT:

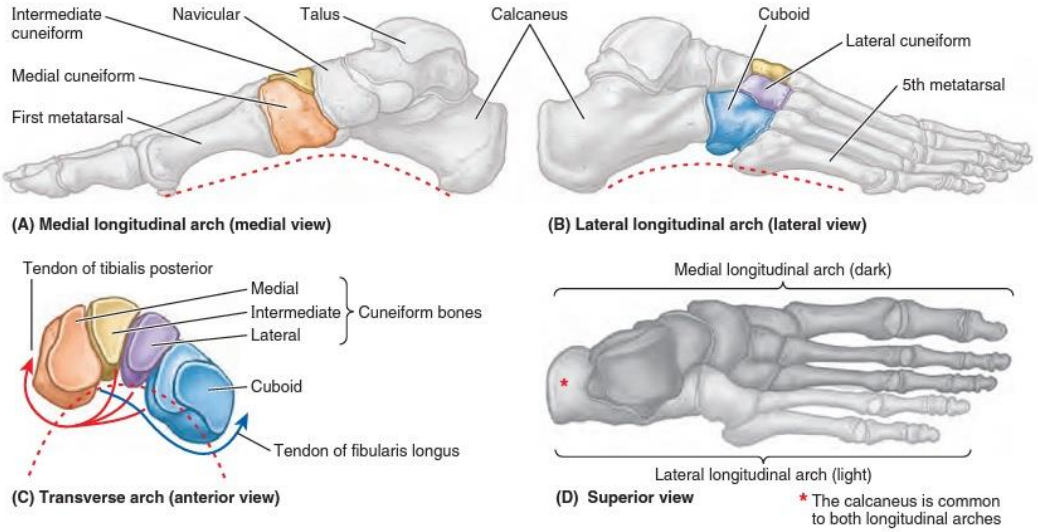
Shape of the united bones

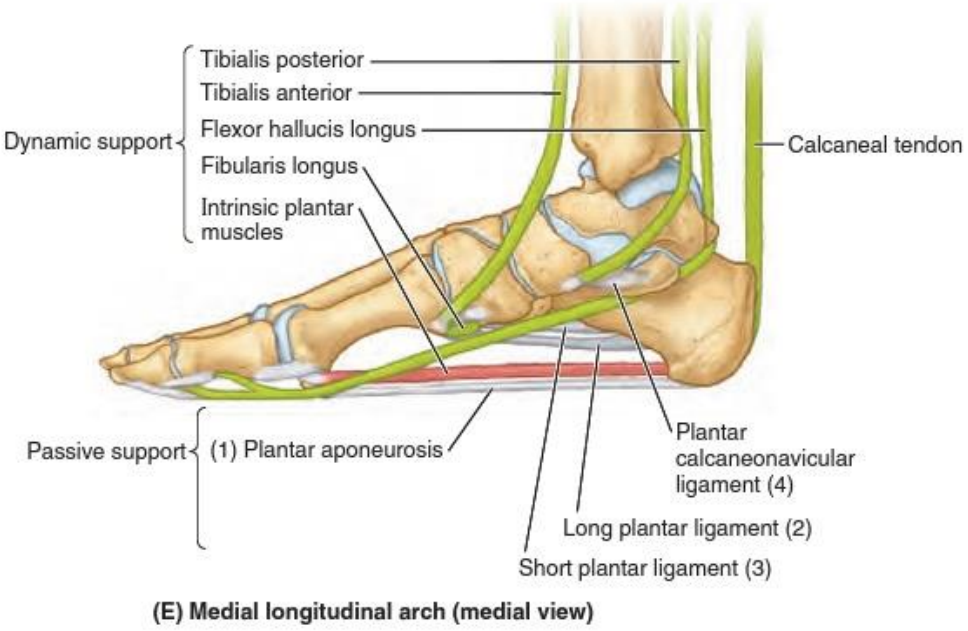
Four successive fibrous tissue layers

- Plantar aponeurosis
- Long plantar ligament
- Plantar calcaneocuboid ligament
- Plantar calcaneonavicular ligament

DYNAMIC FACTORS IN MAINTAINING ARCHES OF FOOT:

- Active bracing of the intrinsic muscles of the foot
- Active contraction of muscles with long tendon
    - Flexor hallucis and digitorum longus
    - Fibularis longus and tibialis posterior


Intermediate cuneiform
Navicular
Talus
Calcaneus
Cuboid
Lateral cuneiform
Medial cuneiform
5th metatarsal
First metatarsal
(A) Medial longitudinal arch (medial view)
(B) Lateral longitudinal arch (lateral view)
Tendon of tibialis posterior
Medial
Intermediate
Lateral
Cuneiform bones
Cuboid
Tendon of fibularis longus
(C) Transverse arch (anterior view)
Medial longitudinal arch (dark)
Lateral longitudinal arch (light)
(D) Superior view
* The calcaneus is common to both longitudinal arches


Dynamic support
Tibialis posterior
Tibialis anterior
Flexor hallucis longus
Fibularis longus
Intrinsic plantar muscles
Calcaneal tendon
Passive support
(1) Plantar aponeurosis
Plantar calcaneonavicular ligament (4)
Long plantar ligament (2)
Short plantar ligament (3)
(E) Medial longitudinal arch (medial view)

ANATOMICAL PRINCIPLES OF SURGICAL INCISIONS:

Some of the anatomic principles one should keep in mind while making incisions in the foot

- Avoid neuro-vascular injuries.
- Avoid incision in weight bearing points.
- Always make liberal counter incision.
- De-roofing should be liberally done
- Always excise the metatarsal head while doing toe amputation for better realignment of the toes.

ULCER

An ulcer is defined as “Any break in the continuity of the epithelium of the skin or mucous membrane”.

It may be caused by molecular death of the surface epithelium or traumatic removal of epithelium

CLASSIFICATION OF ULCER:

I. ACCORDING TO DURATION:

- ACUTE -less than 2 weeks
- CHRONIC – more than 2 weeks

II. CLINICAL:

- **SPREADING ULCER:**

  It is an acute painful ulcer . Edges are inflamed , irregular edematous. Floor is covered with profuse purulent discharge and slough with surrounding edematous skin. Regional lymph nodes are enlarged and often tender.

- **HEALING ULCER :**

  Edges of healing ulcer is sloping and the floor is covered with healthy pink granulation tissue with minimal serous discharge. Regional lymph nodes may or may not be tender and always non-tender. Surrounding skin often does not

show any edema or induration. Three zones are seen in healing ulcer. *Innermost* red granulation tissue ; *middle* bluish growing epithelium zone and the *outermost* whitish fibrosis and scar zone.

- **CALLOUS OR NON HEALING ULCER:**

Pale granulation tissue seen in the floor of the ulcer. Edges and the surrounding skin show induration. Ulcer doesn't show any tendency of healing.

III. PATHOLOGICAL CLASSIFICATION:

- NON SPECIFIC ULCERS:
  - Traumatic
  - Arterial ulcer due to ischemia
  - Venous ulcer eg. Varicose veins
  - Neurogenic ulcer
  - Martorell's ulcer
  - Bazin ulcer
  - Tropical ulcer eg. Vincent's ulcer

- **SPECIFIC ULCERS :**
    - Tuberculous ulcer
    - Syphilitic ulcer
    - Actinomycosis
    - Meleny’s ulcer

- **MALIGNANT ULCERS :**
    - Squamous cell carcinoma
    - Basal cell carcinoma
    - Malignant melanoma

CLASSIFICATION OF WOUNDS

1. RANK AND WAKEFIELD CLASSIFICATION:

- **TIDY WOUNDS:**

Clean wounds like surgical wound without obvious skin loss,wounds caused by sharp instruments

Healing is mainly by primary intention

- **UNTIDY WOUNDS:**

Dirty wounds due to

- Avulsion
- Crushing
- Multiple lacerated wounds
- Burns
- Tearing
- Devitalized tissue

2.BASED ON TYPE OF WOUNDS:

a) Clean incised wounds:- surgical wounds with clear cut edges without skin loss comes in this category

b**) Lacerated wounds:-** wounds with irregular edges , loss of skin

c**) Contusion :-** wounds without skin loss with minimal tissue injury and discolouration

d) Closed blunt injury

e) Hematoma

f) Puncture wounds

g) Gunshot wounds

h) Penetrating injuries

3. BASED ON THE THICKNESS OF THE WOUND:

1)Superficial wounds:- loss of skin epidermis only

2) Partial thickness wound :- loss of epidermis and dermis and only the deep dermis with sweat gland left

3) Full thickness wound :- complete loss of both epidermis and dermis and exposing the subcutaneous tissue.

4) **Deep wounds:-** extend into deep fascia and expose the muscle , tendon or bone

CLASSIFICATION OF SURGICAL WOUNDS:

1)Clean wounds- hernia and cardiovascular surgeries

2)clean contaminated :- bowel , biliary and pancreatic surgeries

3) Contaminated wounds:- accidental wounds and acute abdominal conditions

4) Dirty wounds :- pyocele , abscess drainage , biliary peritonitis

Wounds are classified into two categories – open and closed . The closed wound happens when a blunt force contusion and damages the part of the skin . Closed wounds are as dangerous as an open wound .It is divided into contusion , hematoma , crush injury .

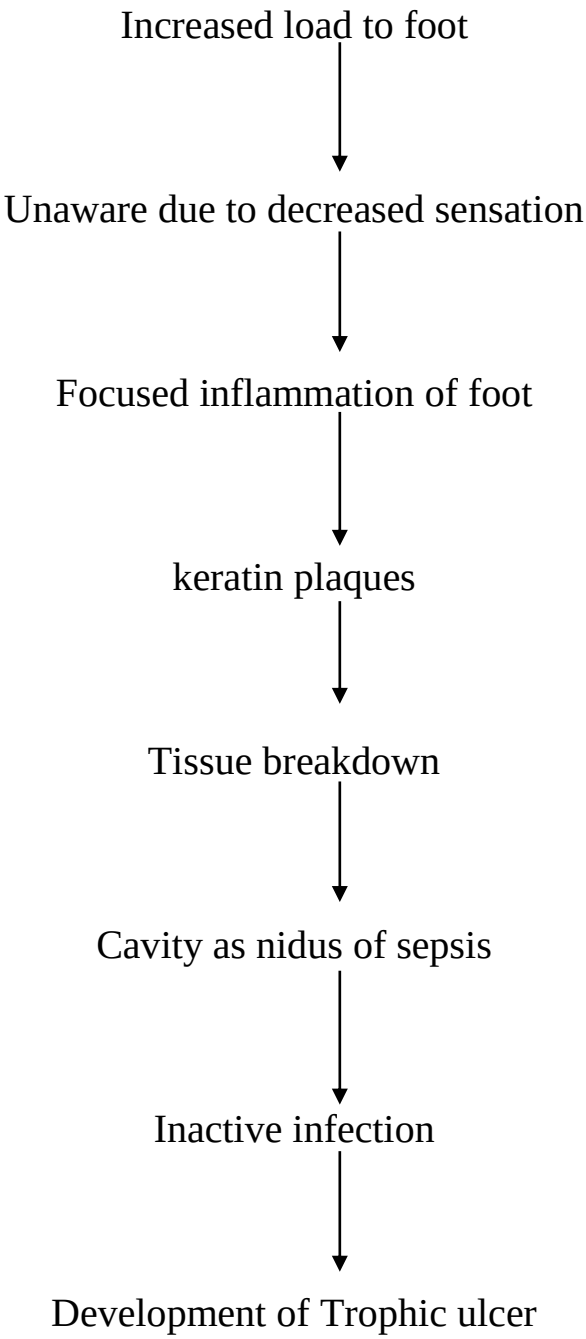
WAGNER classification system of diabetic foot

Grades	Features
0	No open ulcers or may have deformity or cellulitis
1	Ulcer - superficial
2	Ulcer deep to joint capsule or tendon
3	ulcer deep with abscess, joint sepsis or osteomyelitis
4	Local gangrene of foot either fore foot or heel
5	Gangrene invoving the entire foot

TEXAS classification system of diabetic foot

Stages	Grades			
	0	1	2	3
A	Pre or post ulcerative lesions are completely epithelized	Superficial wound are not involving tendon,capsule,or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infected	Infected	Infected	Infected
C	Ischemic	Ischemic	Ischemic	Ischemic
D	Infected and ischemic	Infected and ischemic	Infected and ischemic	Infected and ischemic

NEUROPATHIC ULCER- PATHOPHYSIOLOGY



WOUND HEALING

Wound healing is complicated process to accomplish the functional and anatomical integrity of the disrupted tissues by various mechanisms.

TYPES OF WOUND HEALING:

1) **HEALING BY PRIMARY INTENTION:**

Wounds with clear cut edge with no skin loss such as surgical incision heal by primary intention. Epithelization rate is higher and heals quickly

2) **HEALING BY SECONDARY INTENTION:**

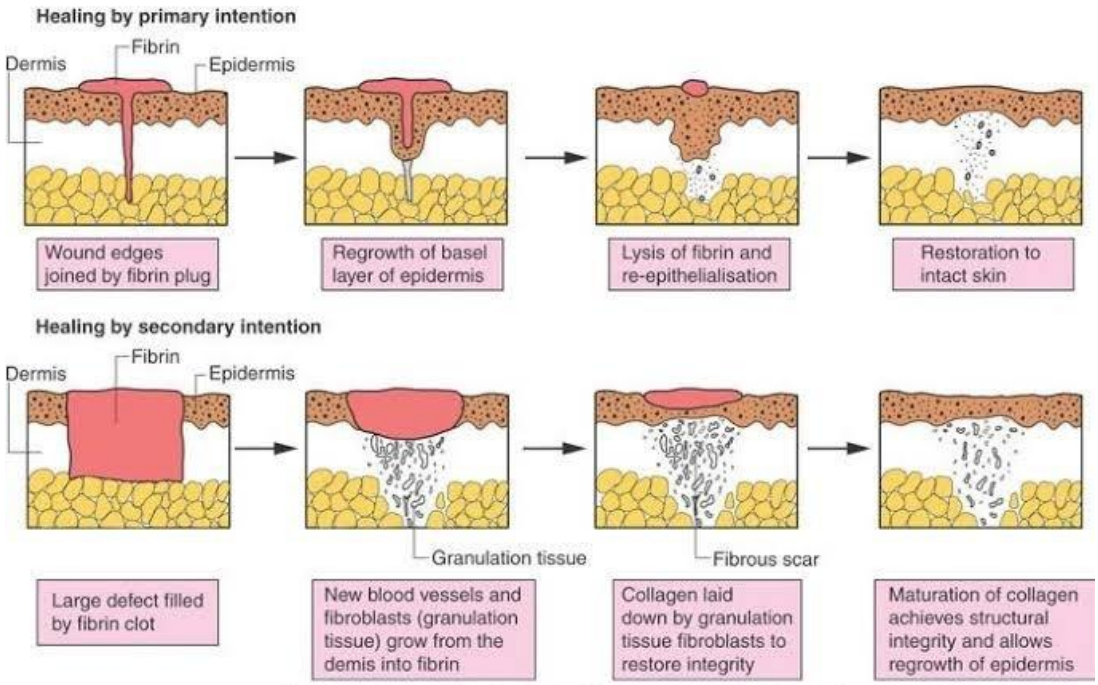
Wounds with large area of skin loss such as contaminated wounds heal by secondary intention . Heals slowly with fibrosis and re-epithelization from the wound edges

3) **TERTIARY INTENTION:**

Wound is initially infected and pus will be present. After regular cleaning and dressing and controlling the infection ,wound closed with sutures or split skin graft

PRIMARY AND SECONDARY HEALING

CHARACTERS	PRIMARY	SECONDARY
CLEANLINESS	CLEAN	NOT CLEAN
INFECTION	NOT INFECTED	INFECTED
SUTURES	USED	NOT USED
MARGINS	SURGICALLY CLEAN	IRREGULAR
HEALING	SMALL GRANULATION TISSUE	LARGE
COMPLICATIONS	NOT FREQUENT	FREQUENT
OUTCOME	LINEAR SCAR	IRREGULAR SCAR



PHASES OF WOUND HEALING

There are four distinct phases of wound healing

- Hemostasis
- Inflammation
- Proliferation
- Tissue remodeling

HEMOSTASIS PHASE:

Hemostasis is the process of the wound closed by clotting. Hemostasis starts when blood leaks out . The first step is blood vessels constrict to restrict the blood flow. Then the platelets stick together to seal the break in wall of the blood vessel. Coagulation occurs and reinforces the platelet plugs with threads of fibrin . The platelets adhere to the endothelial surface within seconds of the rupture of a blood vessel wall epithelium . After that, the fibrin strands begin to adhere in about sixty seconds.

As more fibrin begin to adhere, the blood is transformed from liquid to gel form through the pro-coagulants and the release of prothrombin . The formation of a thrombus keeps the platelets and other blood cells trapped in the wound site.

INFLAMMATORY PHASE:

Inflammation is the second stage of wound healing. It begins within 6-8 hrs and lasts for 5-7 days .It begins right after injury when the injured blood vessels leak transudate causing localized swelling. Inflammation controls both the bleeding and also prevents infection. Macrophages release FGF which is responsible for the angiogenesis. Polymorphonuclear leukocytes secretes inflammatory mediators and free radicals after 48 hours . These mediators remove the foreign body , clots and bacteria.

PROLIFERATIVE PHASE:

The proliferative phase starts after 7 days and lasts for more than 6 wks. Fibroblast secrets collagen and glycosamines. Proliferation of the vascular endothelial cells occurs at the wound edges by the cytokines and growth factors with the help of fibroblasts. Hydroxylysine and hydroxyproline secreted by various enzymes with the help of vit C , iron and alpha ketoglutarate.

Proliferative phase has 3 sub-phases

- Neovascularization

- Fibroplasias
- Collagen and re-epithelization

i) Neovascularisation:

It is the process of regeneration of new blood vessels. It concurrently occurs with the endothelial cell migration and proliferation of fibroblast. Transforming growth factor – β , fibroblast growth factor and VEGF (vascular endothelial growth factor) facilitates the endothelial cell elongation to cause neovascularization in the granulation tissues. It is promoted by hypoxic conditions , presence of lactic acid and reduced oxygen environment.

At the end of this phase ,when the tissues are perfused with sufficient nutrients the blood vessels no longer needed will undergo apoptosis

ii) **Fibroplasias and tissue formation:**

Fibroblasts are important component in the granulation tissue that appears after inflammatory phase. When the angiogenesis stops newly formed endothelial cell , myofibroblast and ECM grows continuously until the wound bed covers completely.

iii) Collagen deposition :

Fibroblasts are the maximum contributor of collagen synthesis and deposition and increases the strength of the wound by facilitating the angiogenesis and differentiation of the connective tissues. The type III collagen and fibronectin are synthesized between 10 hours and 3 days after injury and the deposition of collagen peaks between one to three weeks depends on the wound size.

REMODELLING:

This phase starts by 6 weeks and lasts for upto 2 years . cross linking of the collagen takes place in this phase laeding to the maturation of the collagen responsible for the tensile strength of the wound. usually synthesis did not occur after 42 days of wound healing It has 2 sub phases

- **Epithelization**
- **Contraction**

i)Epithelization:

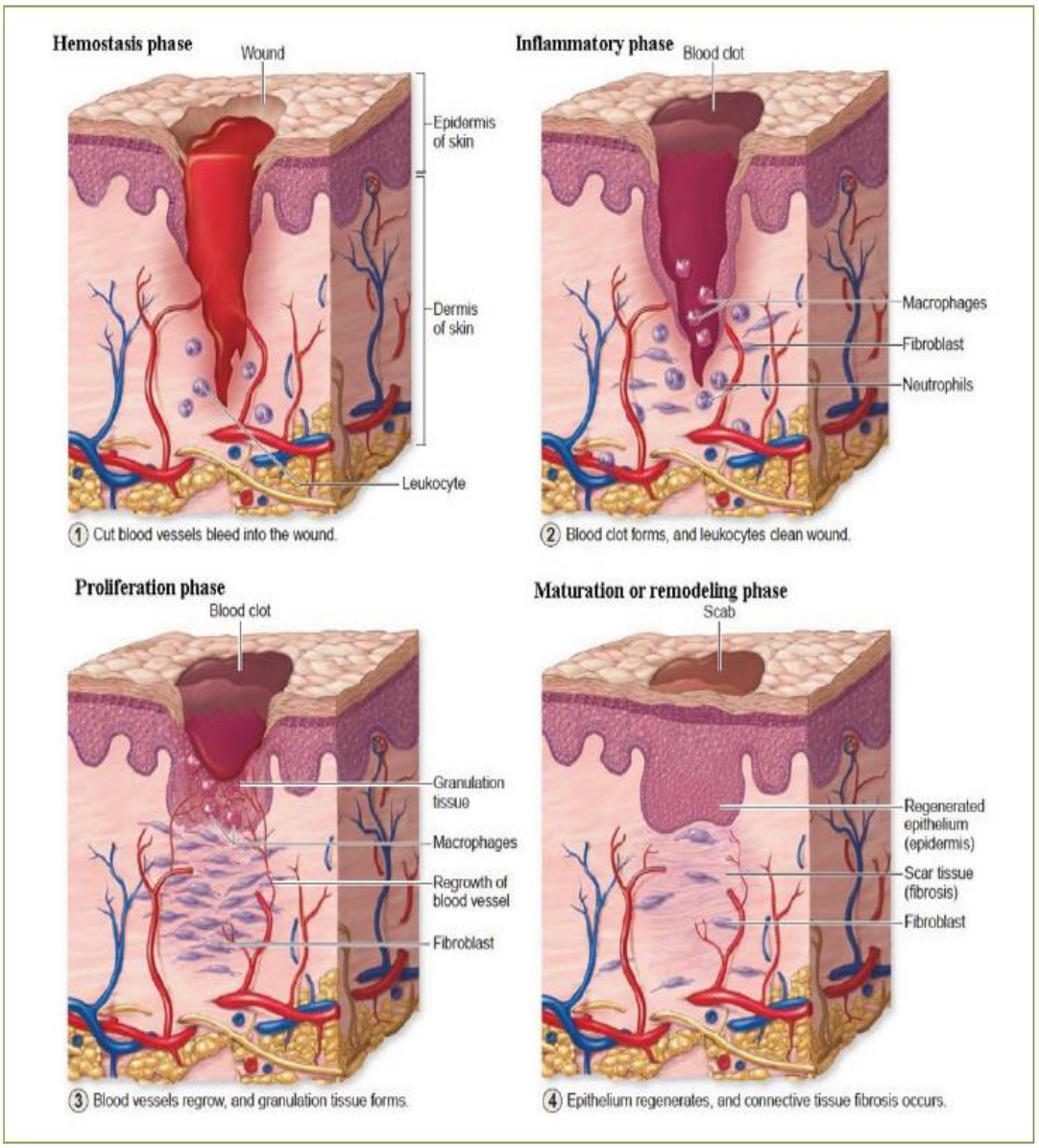
Epithelization occurs after formation of granulation tissue in open wound that allows the epithelial cells and keratinocytes to migrate across the newly formed granulation tissue and wound

surface area. keratinocytes alters their shape and become flat and elongated morphology and thus the cells forms cellular process by actin and lamellipodia. This migration is usually mediated by lack of contact inhibition and nitric oxide etc. they utilize the fibrin to move across the wound site. The epithelial cells formed at the wound edge act as a base for keratinocyte proliferation. Keratinocyte migration will continue cells from other wound edges meet to form contact inhibition which stops migration.

ii) Wound contraction:

contracture is the process of dimunition of the wound surface area that occurs by the centripetal movement of the wound edge towards centre. It usually starts by approximately 4-5 days after wounding at an rate of 0.6-0.7 mm / day depending on the type of tissue and wound. This contraction is brought about by myofibroblast interaction with the ECM. Prolonged contraction leads to skin and muscle deformity and functional disability.

PHASES OF WOUND HEALING



FACTORS AFFECTING WOUND HEALING

LOCAL FACTORS	SYSTEMIC FACTORS
Hypoxia	Diabetes mellitus
Repeated trauma	Anemia
High bacterial burden	Malnutrition
High metalloproteinases	Immunodeficiency
Growth factor deficiency	Immunosupressants
Cellular senescence	Age
Irradiation	Obesity
Necrotic tissue burden	Smoking
Excessive exudates	
Corrupt ECM	

COMPLICATIONS OF WOUND HEALING

1. **Deficient scar formation:** due to improper formation of the granulation tissue

2. **Incisional hernias :** Dehiscence of scar results from increased pressure from within . Abdominal dehiscence carries a mortality rate of upto 30 %

3. **Ulceration:** wound ulcerate because of the inadequate blood supply as in the case of leg ulcers with atherosclerosis or varicose veins. Repeated trauma due to impaired sensation leads to trophic or neuropathic ulcer as in Diabetes or leprosy.

4. **Excessive scar formation :** An exuberant granulation tissue formation results in hypertrophic scar or keloid formation. Both these scars exhibit abundant irregular collagen bundles with more fibroblast and capillaries than the scar of same age.

5. **Excessive contraction :** an exaggeration in the process of contraction leads to severe wound deformity , for eg . Dupuytren's contracture , plantar contracture and the peyronie's disease of penis. Contracture may compromise joint movements . contracture in the esophagus or intestine causes intestinal obstruction .

CAUSES FOR THE FORMATION OF NON HEALING ULCER

- Repeated infections
- Decreased blood supply
- Hypoxia
- Loss of sensation
- Malignancy
- Surrounding area edema
- Systemic illness like TB , Diabetes mellitus
- Underlying osteomyelitis
- Fibrosis

MANAGEMENT OF THE DIABETIC FOOT:

The foot ulcers in diabetics are generally not a non healing ulcers but they are mal treated ulcers.

(a) WAGNER's grade 0 foot:

This grade includes the patients with apparently normal foot but varying degree of neuropathy or joint deformities. They donot have an ulcer or infection but are potentially at risk. They need regular assessment annually. Neuropathy must be examined during each assessment. The best way to prevent neuropathy or delay it is to keep glycemic control.

Assessment of vascular status is al mandatory. Absent pulses in the foot even in the absence of "rest pain or claudication" indicates a signicant vascular disease and such patients may be ideal candidates for the vascular reconstruction or angioplasty. The "at risk" patients have elevated pressure over some points on the sole of the foot. They need to wear appropriate footwear .Charcot's foot need custom shoes. Regular trimming of the callus is needed.

(b) WAGNER's grade 1 foot:

These patients have presented with either superficial ulcer. Ulcer occurs either with repetitive low or high pressure at any pressure point on sole during walking.

Relief of pressure is mainstay of ulcer treatment. An ulcer will not heal if the patient walks with the ulcer. The various methods of "off-loading" devices are used. the appropriate management of vascular disease is needed as in grade 0. Infection needs debridement and antibiotics as appropriate.

(**c) WAGNER grade 2 and 3 foot:**

patients who have deep ulcer with or without complications like osteomyelitis or abscess. These patients requires initial aggressive debridement. Osteomyelitis must be managed by debridement or excision of the infected bone.

The patient requires follow up for long duration to advise appropriate foot wear and education regarding foot care to prevent formation of further ulcer.

(d)WAGNER grade 4 and 5 foot:

They require minor or major amputation. Always there is concomitant vascular occlusive disease. These patients therefore need appropriate vascular reconstructions. After the ulcer heals patient needs to wear special footwear for the ipsilateral and contra-lateral foot. For the major amputees, prosthetic devices should be fitted in order to mobilize the patient.

Principles of medical management:

1. Pus from ulcers should be sent for pus culture and sensitivity.

2. Careful monitoring of random and fasting blood glucose levels.

3. Appropriate antidiabetic drugs measures – either insulin or oral hypoglycemic agents.

4. Broad spectrum antibiotics covering aerobic and anaerobic organisms should be started at the onset and change to other antibiotics depending on the culture and sensitivity report.

Principles of the surgical management of diabetic ulcer:

1. Early diagnosis of ulcer and prompt intervention.

2. Correction blood glucose.

3. Complete rest to the injured area.

4. Careful and complete debridement and drainage of all involved areas at the initial visit itself.

5. Appropriate antibiotics coverage.

6. regular wound care and dressings.

7. Appropriate vascular reconstruction if needed .

8. Careful follow up with podiatric appliances and modified footwear.

MANAGEMENT OF CHRONIC ULCER

The ideal wound management plan should be less infectious, cost effective , low exchange of water vapour , allows healing without delay and avoid damaging the newly formed tissues. Some disease such as atherosclerosis, DM and conditions such as anemia , malnutrition and local infections cause delay in wound healing. Supplementation of appropriate diet and treatment of underlying disease is necessary to assist in faster wound healing. Infection is the most common cause for death in the burns injury and accounts for 50 % of the hospital deaths in burns . so it is essential that the wound management should be done with sterile and aeptic conditions.

INVESTIGATIONS:

- Basic blood investigations – HB% , blood sugar , creatinine and urea
- Serum proteins and electrolytes
- Pus culture and sensitivity
- Local parts X – ray to rule out periostitis and osteomyelitis

- In suspected tuberculosis – Chest x –ray and Mantoux
- Edge wedge biopsy if suspicious of malignancy
- Venous and the arterial Doppler study – to rule out venous and arterial insufficiency

ASSESSMENT OF THE ULCER :

- Cause of the ulcer should be identified initially
- Systemic examination should be done to assess the sensation , movements , peripheral pulses and lymph node status.
- Parameters assessed:
    - Location
    - Size
    - Signs of infection
    - Surrounding skin – colour and temperature
    - Wound edges- induration or maceration
    - Wound bed – slough , necrosis , granulation.

MANAGEMENT OF CHRONIC ULCER:

Adequate debridement of all necrotic tissue (eschar , slough) is essential before adequate assessment and staging of the ulcer. There are various methods of debridement which includes sharp surgical debridement , mechanical debridement , enzymatic and autolytic debridement. It is continuum from flushing away necrotic debris with low pressure irrigation upto wide excision.

1.Sharp surgical debridement

The most efficacious method of debridement is sharp surgical debridement. Debridement of ulcer base upto bleeding is ideal method of debridement for the patient with non healing ulcer.

2.Mechanical debridement

Mechanical debridement can be accomplished by wet to dry gauze dressings , pulsatile lavage ,irrigation or a whirl pool.

3.Autolytic debridement

Autolytic debridement with moist dressings is selective and it liquefies the slough and eschar and also it promotes the granulation tissue formation.

4.Enzymatic debridement

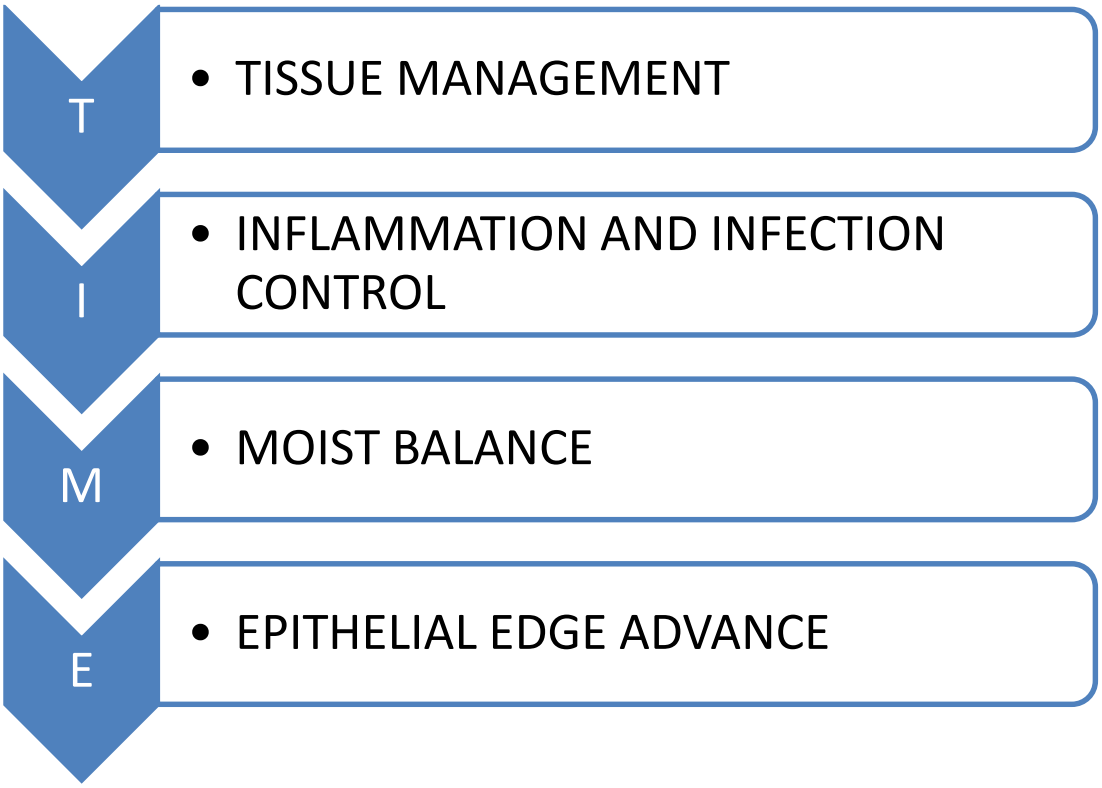
Enzymes such as collagenase , papain, urea has been used as debridment agents for eschar and slough. They are costly and labour intensive method of debridement.

5. Antibiotic

The use of double or triple antibiotics is justified. The antibiotics used must have broad spectrum coverage of gram positive, negative and also anaerobic organisms.

TIME APPROACH FOR CHRONIC NON HEALING ULCER

FALANGA (2004) utilised the work of SCHULTZER et al (2003) to develop a framework called TIME to provide the comprehensive approach for wound care. The TIME principle guidelines helps in approach for the best wound care.



CLASSIFICATION OF DRESSING :

Dressings are classified into two major categories according to the usage

1.**long term application or skin substitutes:**

Further subdivided into :

- **Temporary :** it is applied onto the fresh partial thickness wound until complete healing of the wound is ensured
- **Semi permanent –** it is applied on the full thickness wounds until the auto-grafting of the wounds.

2.**Short term application :**

Dressing usually requires replacement at regular intervals

It is further classified into conventional , synthetic and biological based on the type of material used in the dressing

A.Conventional:

B. Synthetic :

i) Films

ii) Composite

iii) Foams and spray dressing

C. Biological :

i) Allograft

ii) Xenograft

iii) Collagen dressing

Within each **category** , dressing are sub classified into

1. **Primary -** dressing contact with the ulcer bed
2. **Secondary-** dressing which covers the primary dressing
3. **Island –** dressing constructed with adhesive portion outside and a central absorbent

MANAGEMENT OF INDOLENT NON HEALING ULCERS

a. Collagen

b. Silver colloid dressing

c. dressings with Growth factors

Growth factors are derived from the platelets and bioengineered tissues or by the recombinant techniques.

NEGATIVE PRESSURE WOUND THERAPY

Vacuum assisted closure dressing(VAC), also called as negative pressure wound therapy (NPWT) or Micro deformational wound therapy , brought a revolution in wound care since past 20 years. This method was first coined by **Fleischmann *et al*.** in 1993.

In this era of modern wound care, negative pressure therapy has been routinely used for treatment of wounds and become the integral part of the treatment plan of the chronic non healing ulcers. It is used in acute, chronic or complex wounds and has been proven more efficacious and promotes for faster healing than the conventional dressing.

The trademark VAC therapy belongs to KCI VAC needed a sophisticated equipment, specialized foam and drain and also trained person for the application and maintenance, which is possible only at high cost settings.

In our study, we used the easily available materials in low resource setting such as gauze , glove to make the negative pressure and making the cost effective manner.

FOUR PRIMARY MECHANISMS:

1. Macro deformation

2. Microdeformation near

3. Removal of excess exudates

4. Optimisation of the wound

1. MACRODEFORMATION

It refers to the decrease in the wound surface by shrinkage of sponge and action of the centripetal forces over the wound surface. Due to the inherent tension present in the dermis near the wound and the underlying attachments of different wounds over the different sites contract wound to different extent.

2. MICRODEFORMATION:

The vaccum transmitted through the interface material acting over undulated surface of wound produces changes occur in micro to millimeter range scale. The mechanical forces are transmitted to the every cell through the ECM and lead to the cell deformation causing modification in the cell function for adapting the stress.

3. REMOVAL OF EXCESS FLUID

Excess fluid in ECF leads to edema and the deprivement leads to signs of dehydration. This compartment is drained by the lymphatics ; abnormality may lead to lymphedema. Excess of fluid cause delay in the wound healing due to compressive effect over the tissues. Removal of the excess fluid from the wound will decrease the compression effect over the microvasculature and thereby promoting the perfusion and wound healing.

CONTRAINDICATIONS:

- Untreated osteomyelitis
- Non enteric fistulas
- Necrotic tissues along with eschar
- Exposed blood vessels
- Malignancy
- Exposed nerves
- Anastamotic sites
- Exposed internal organs

AIM & OBJECTIVE

To compare the efficacy of the modified vacuum assisted closure (VAC) dressing using gloves and available resources in a low resource setting with routine Povidine iodine dressing in wound healing in patients who are admitted in GRH,MADURAI

MATERIALS AND METHODS

DESIGN OF THE STUDY: PROSPECTIVE STUDY

STUDY **PERIOD** **: 2019**

SAMPLE SIZE **: 100**

STUDY PLACE **: GRH MADURAI**

INCLUSION CRITERIA:

- Patients more than 25 years of age
- Diabetic ulcers in the extremities
- Non healing ulcers in the extremities
- Amputation stump ulcer

EXCLUSION CRITERIA

- Malignant wounds
- Wounds with underlying osteomyelitis
- Wounds with sinus or cavity
- Wounds with unstable fracture
- Wounds with loose fragment of bone

- Larger wound surface
- Wounds with Exposed blood vessels
- Patients on anticoagulation therapy

METHOD OF COLLECTION OF DATA

Detailed history

Clinical Examination

Dimensions of the ulcer

Rate of granulation tissue

Duration of hospital stay

METHODOLOGY:

- wound debridement
- The wound base was covered with gauze piece placed in two layers.
- A tube with adequate fenestrations depending on the size of the wound placed in between the two layers of gauze.

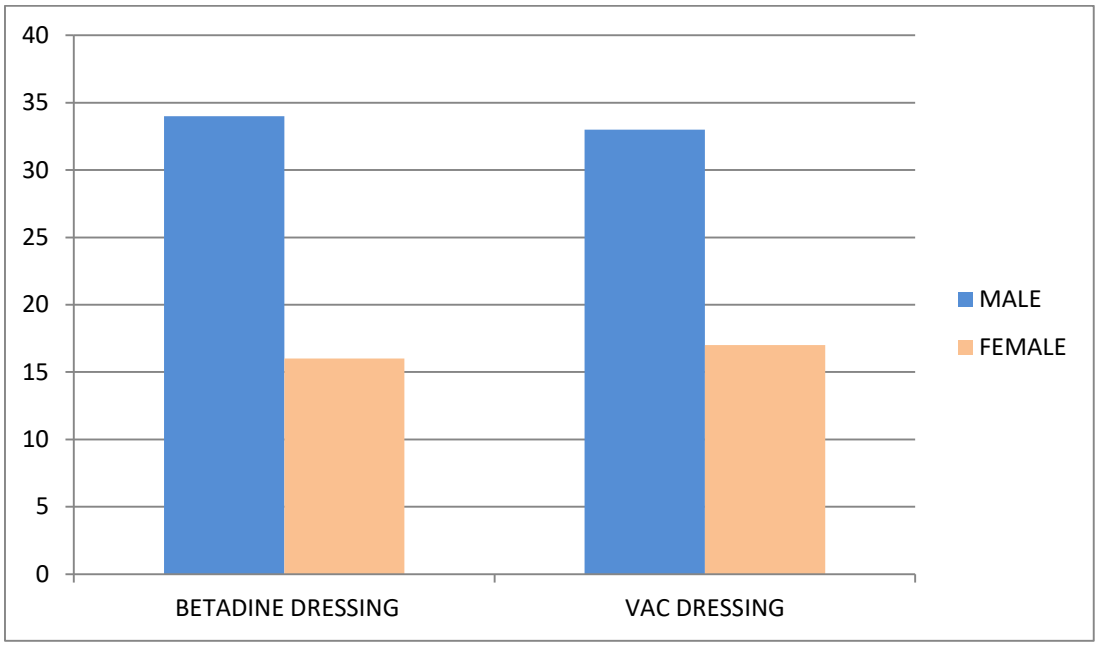
- The gauze layer is held in place over the wound by applying thin Opsite.
- An appropriate sized sterile surgical glove is taken and inserted over the wound covering the extremity
- The tube is brought out from the edge of the glove.
- A piece of Opsite is applied covering entire margin of glove circumferentially.
- Exit site of tube through the glove is fashioned like T-tailing using opsite strip to prevent air leakage
- End of the tube connected to Romovac device or 50 cc syringe and suction is applied

Total ulcer surface area measured initially and the reduction in the surface area before grafting measured. Area of granulation tissue covering the ulcer also measured using butter paper. After grafting on the post op day 5 graft uptake was measured in percentage. Total duration of the hospital stay was noted . pain in the each patient was measured using visual analogue scale.

OBSERVATIONS AND RESULTS

The 100 patients admitted in our GRH selected for the study were divided into two equal and comparable groups. Patients subjected to modified VAC dressing using gloves were classified under study group and those who underwent conventional povidone iodine wound dressing were classified under control group.

Graph: Sex wise distribution of patients.



	MALE	FEMALE
BETADINE DRESSING	34	16
VAC DRESSING	33	17

TABLE: Age wise distribution of patients

AGE GROUP (yrs)	31-40	41-50	51-60	61-70	71-80
BETADINE	7	11	17	10	5
VAC	7	14	16	8	5
TOTAL	14	25	33	18	10

Mean age of Betadine group is 54.42

Mean age of VAC DRESSING group is 54.7

P value is 0.90.Not significant.

GRAPH : Age wise distribution of patients

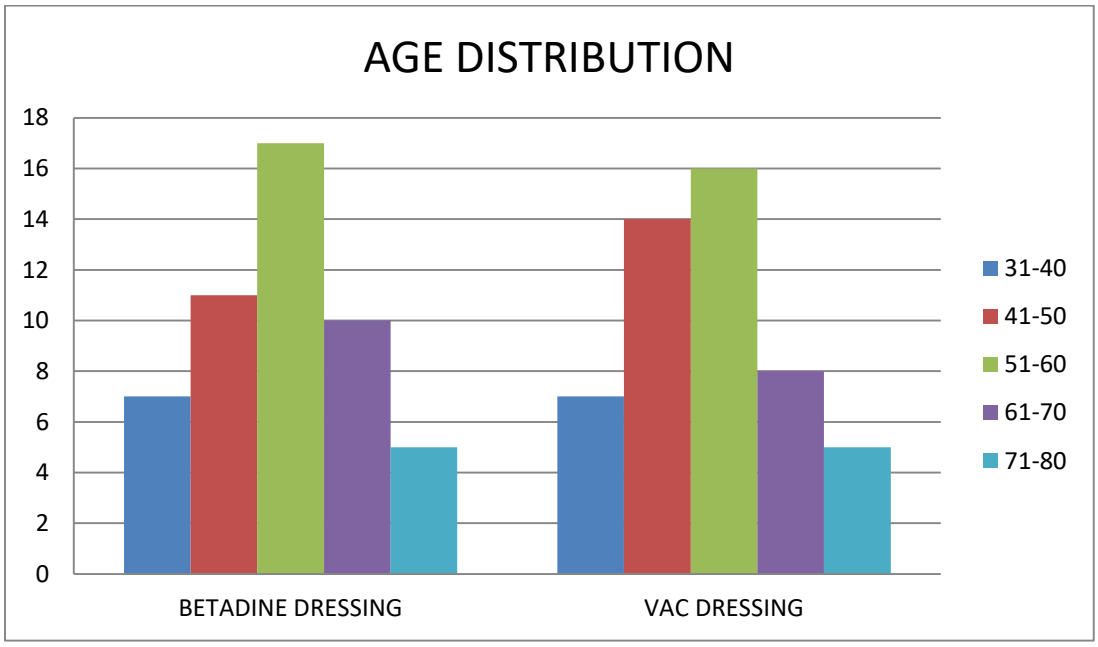


TABLE: ULCER SURFACE AREA

GROUP	NO.OF PTS	MEAN	STD. DEVIATION	t VALUE	p VALUE
BETADINE	50	38.74	5.73	1.82	0.07
VAC DRESSING	50	40.41	2.85		

The mean ulcer area in control group is **38.74+5.73(SD)cm^2** and in the study group is **40.41+2.85(SD)cm^2** . The ulcer area was measured by using tissue paper.

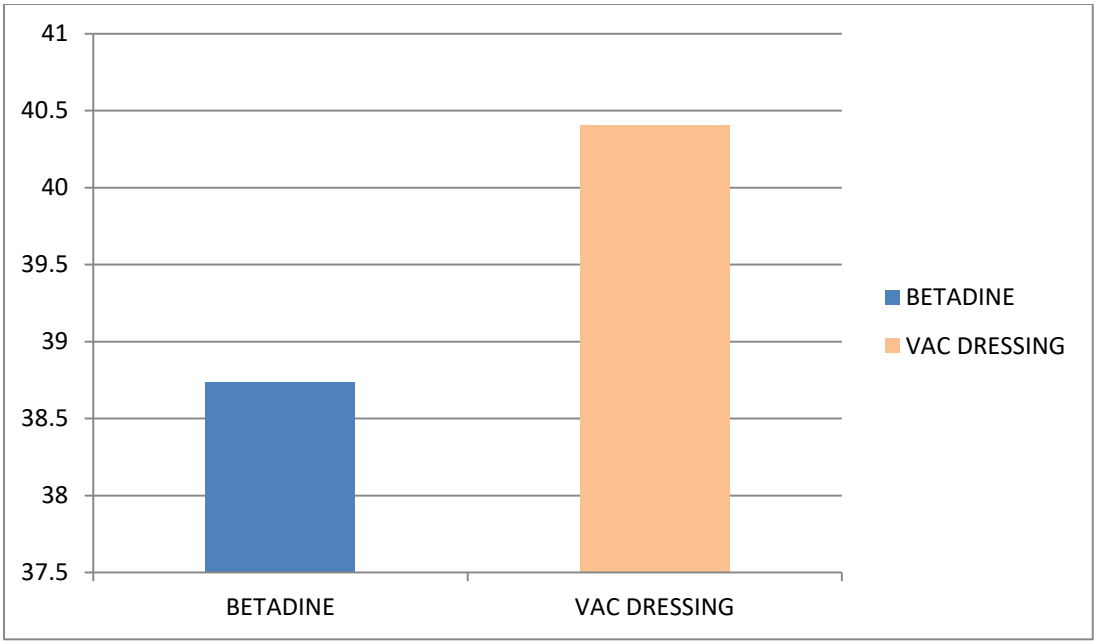
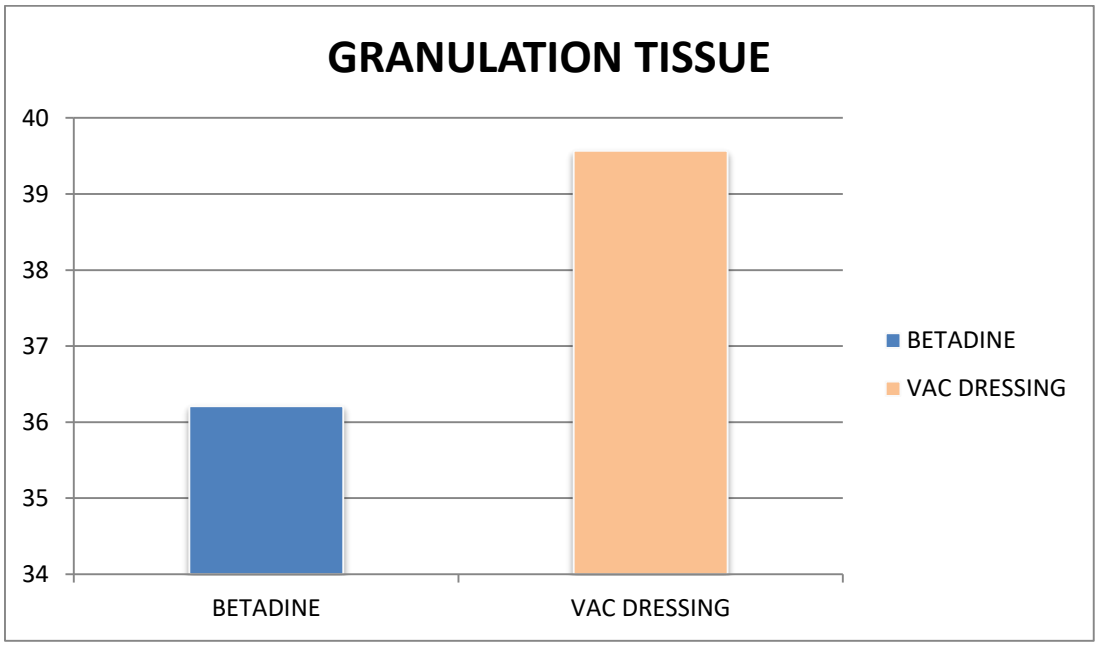


TABLE: RATE OF GRANULATION TISSUE FORMATION

GROUP		NO.	MEAN	STD. DEVIATION	t VALUE	p VALUE
GRAN TISSUE	BETADINE	50	36.21	5.99	3.58	0.0005
	VAC DRESSING	50	39.57	2.69		

The mean rate of granulation tissue formation in Betadine group is **36.21+5.99(SD)** of total ulcer surface area and in VAC DRESSING is **39.57+2.69(SD)** of total ulcer surface area.

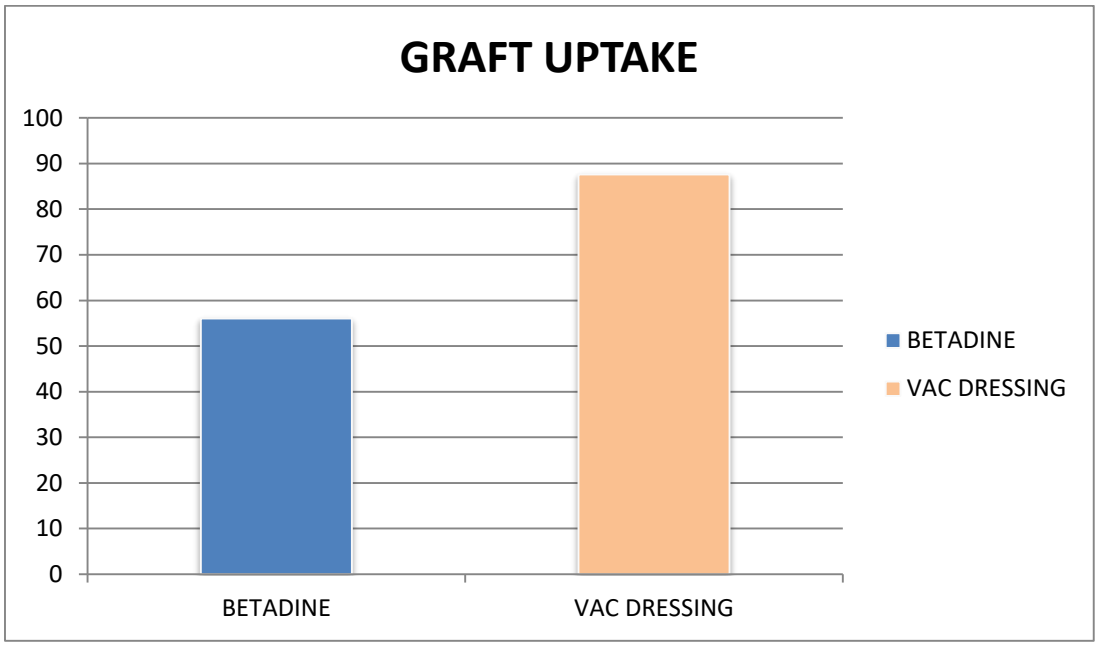


The mean rate of granulation tissue formation in Betadine group is **36.21+5.99(SD)** of total ulcer surface area and in VAC DRESSING is **39.57+2.69(SD)** of total ulcer surface area.

TABLE: GRAFT UPTAKE AS PERCENTAGE OF ULCER SURFACE AREA

GROUP		N	MEAN	SD	t VALUE	p value
SSG	**BETADINE**	50	56.06	19.05	10.9	0.0000
	VAC DRESSING	50	87.6	6.62		

Assessment of graft uptake was done at the end of POD 5 as percentage of ulcer surface area. The mean graft uptake in the study group is **87.6%** and in the control group is 56.0**6 %**

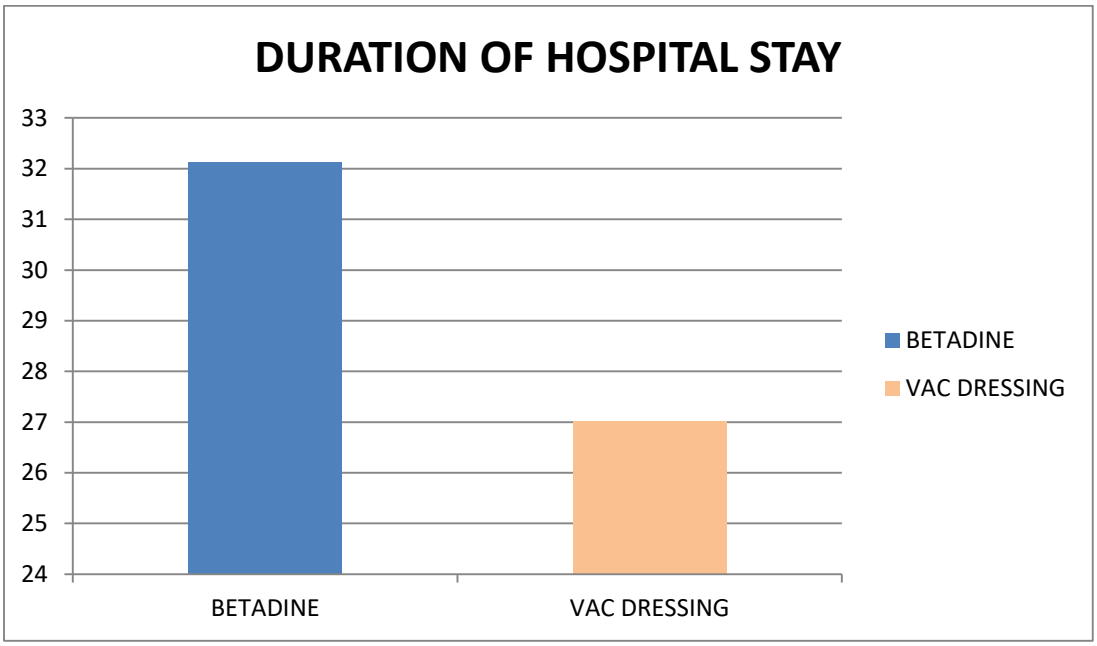


Assessment of graft uptake was done at the end of POD 5 as percentage of ulcer surface area. The mean graft uptake in the study group is **87.6%** and in the control group is 56.0**6 %**

TABLE: DURATION OF HOSPITAL STAY

GROUP		N	MEAN	STD DEVIATION	t VALUE	p VALUE
NO OF DAYS	BETADINE	50	32.12	6.07	4.87	0.000004
	VAC DRESSING	50	27.02	4.09		

The mean duration of hospital stay in the vacuum group was 27.02 days and in the povidone iodine dressing group was 32.12 and the p value was ($p<0.000004$) which is highly significant.

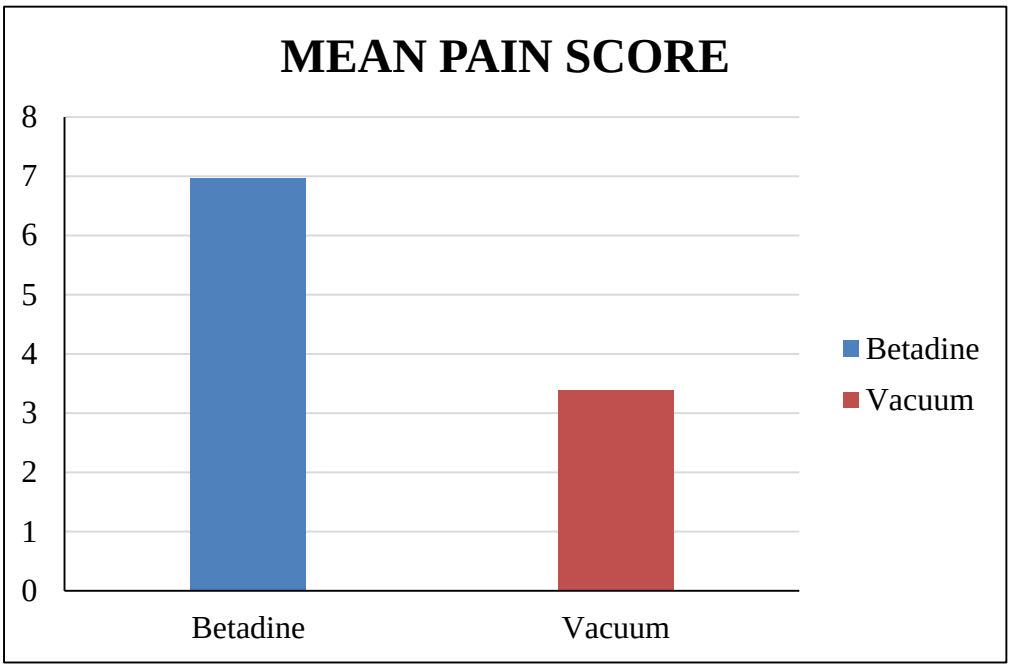


The mean duration of hospital stay in the vacuum group was 27.02 days and in the povidone iodine dressing group was 32.12 and the p value was ($p<0.000004$) which is highly significant.

PAIN SCORING

Average pain score in the range of 0 to 10 was 6.96 in the conventional betadine dressing and it was 3.38 in the study group. P< 0.001 which is significant reduction in the pain score.

PAIN	TEST	CONTROL
MEAN	3.38	6.96
SD	1.24	1.41
P VALUE	<0.001	



Average pain score in the range of 0 to 10 was 6.96 in the conventional betadine dressing and it was 3.38 in the study group. P< 0.001 which is significant reduction in the pain score.

The main postoperative parameters noted in the study and control groups:

- Wound size
- Contracture
- Pain
- Infection

All these parameters are less in the study group when compared to the control group.

ANALYSIS OF DATA:

The number of patients studied was 100 and are randomly divided into study (50) and control group (50).both the study and control group were matched regarding their age ,sex and there was no significant difference between the two groups with respect to age and sex.

The average pain score in the range of 0-10 was 3.38 in the vacuum dressing and it was 6.96 in the povidone iodine dressing group.

The mean duration of hospital stay in the vacuum group was 27.02 days and in the povidone iodine dressing group was 32.12 and the p value was ($p<0.000004$) which is highly significant.

The mean split skin graft uptake in the vacuum dressing group was 87.6% and in the povidone dressing group is 56.06% and the p value showed highly significant difference in split skin graft uptake ($p<0.0000$).

The mean rate of granulation tissue formation in povidone group is 36.21 of total ulcer surface area and in vacuum dressing group is 39.57 and the p value was ($p<0.0005$) which is highly significant

CLINICAL PICTURES

CLINICAL PICTURE

CLINICAL PICTURE

CONCLUSION

- Modified VAC dressing significantly reduces the size of ulcer.
- Modified VAC dressing improves the rate of granulation tissue formation.
- Modified VAC dressing improves SSG uptake also.
- Modified VAC dressing reduces the duration of stay at the hospital.
- Patients undergoing Modified VAC dressing feels lesser amount of pain when compared with the patients undergoing conventional wound dressing.
- Modified VAC dressing minimizes the blood loss also.

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PROFOMA

Name of the patient:

Age/sex:

IP No:

D-O-A:

D-O-D:

Duration of hospital stay:

Diabetic status:

Other co-morbidities

Alcoholic/ smoker:

History of presenting illness:

Significant past history:

Examination of ulcer:

Number

Site:

Size:

Shape:

Margin/base /floor:

Surrounding skin status:

Feautures of malignancy:

Induration :

Vascular status:

Skin/nail :

Palapation of the peripheral Pulses:

Examination of the regional lymph nodes:

Investigations:

Basic blood investigations:

Hb% / TC /DC /ESR:

Random Blood sugar:

FBS/PPBS(if diabetic):

Blood urea / Sr creatinine:

Chest xray :

ECG:

Arterial and venous Doppler study:

X ray of the local part:

Viral markers:

Pus culture and sensitivity:

Treatment:

Antibiotics :

Insulin dosage / OHA (if DM):

Treatment before Modified VAC dressing:

Modified VAC dressing:

Date of application:

Date of removal:

No of dressings:

Treatment After Modified VAC dressing:

Granulation tissue:

Exudates:

Pain scoring (VAS scale)

Plan after Modified VAC dressing:

MASTER CHART

S No	NAME	AGE	SEX	IP No	TYPE OF DRESSING	ULCER SURFACE AREA	GRANULATION SURFACE AREA	SSG UPTAKE	PAIN SCORE	DURATION OF HOSPITAL STAY
1	KARUPPASAMY	40	M	40711	BET	40.5	37.2	45	5	34
2	GOPAL	51	M	31228	BET	42.5	38	30	7	37
3	RAJALAKSHMI	68	F	32622	BET	44.6	41.2	42	8	32
4	CHELLAMMAL	62	F	33722	BET	42.7	41.2	42	9	28
5	MOHAMMED IBRAHIM	50	M	36282	BET	44.6	42.7	39	8	38
6	RAJESH	56	M	34282	BET	41.9	40.2	48	7	30
7	JEYARAJ	52	M	11286	BET	40.9	39.2	42	6	32
8	RAMAR	43	M	12682	BET	38.6	37	39	7	35
9	SELVARAJ	56	M	13928	BET	36.7	35.2	29	5	28
10	PONNAMMAL	60	F	14682	BET	35.4	32.4	21	6	28
11	PANDIYAMMAL	72	F	14662	BET	36.4	33.2	32	6	32
12	RAGURAMAN	40	M	16282	BET	32.2	29.6	43	8	28
13	MUTHU	56	M	15923	BET	42.6	40.1	42	9	26
14	SARAVANAN	50	M	16283	BET	39.7	37.7	40	9	30
15	PANDI	52	M	14862	BET	44.7	43.4	42	6	31
16	DOGLOUS	62	M	15932	BET	38.6	34.5	36	7	32
17	JESURAJA	70	M	17286	BET	37.8	36.2	34	6	36
18	ANSARI	55	M	24216	BET	30.3	28.6	68	5	40
19	AROCKIYASAMY	55	M	32962	BET	29	26.3	28	6	24
20	CHANDRASEKAR	32	M	36282	BET	32.9	30.1	40	7	30
21	KENNETH	40	M	44028	BET	40	37.6	88	9	28
22	JEGANATHAN	42	M	49628	BET	38	34.5	68	8	26
23	FATHIMA BEGAM	62	F	45326	BET	45.6	41.7	76	9	42
24	JANAKI	75	F	32374	BET	43.2	39.6	72	8	24
25	AMMASI	60	M	32962	BET	28.6	27.3	90	7	27
26	KANNAMMAL	72	F	32419	BET	27.2	24.3	92	6	23
27	SELVI	79	F	32454	BET	32.5	30.1	45	7	30
28	BABU	42	M	56286	BET	29.5	26.2	80	5	28
29	KAMALADEVI	70	F	32452	BET	25.3	23.6	92	4	30
30	ANDIRASU	68	M	32334	BET	40.2	36.2	86	3	45
31	GANESAN	79	M	35692	BET	33.7	30.3	84	7	28
32	VEERAPPAN	45	M	54286	BET	45.3	42.6	55	8	24
33	IRULANDI	62	M	56284	BET	30.9	22.4	54	9	32
34	PERIYASAMY	55	M	59322	BET	52.8	46.4	52	6	38
35	JOSEPH	60	M	59111	BET	42.8	41.8	74	7	32

36	VAHITHA	54	F	57126	BET	42.1	41.5	70	8	32
37	SURESH	35	M	58282	BET	38.7	37.1	76	9	30
38	PANDISELVAM	37	M	55396	BET	39.6	37.1	57	7	35
39	MARIYAPPAN	42	M	60962	BET	41.2	40.6	62	6	32
40	CHITTAN	45	M	70132	BET	43.2	42.1	40	5	42
41	MUTHUPANDI	50	M	61146	BET	50.2	49.6	64	7	40
42	MURUGESHWARI	56	F	70411	BET	32.6	30.2	72	7	34
43	JOHN PETER	58	M	69262	BET	43.6	40.7	66	8	30
44	SEKAR	54	M	73211	BET	41.2	40.6	50	6	26
45	SOMASUNDAR	62	M	66636	BET	38.6	33.8	52	7	28
46	NAGALAKSHMI	70	F	70212	BET	36.5	34.2	80	8	24
47	SAKTHIVEL	43	M	71396	BET	40.8	38.9	72	9	32
48	MUTHUSELVI	38	F	76928	BET	38.7	36.8	60	8	36
49	ZAKIR HUSSAIN	42	M	39286	BET	41.2	40.2	44	7	45
50	MUNEESWARI	56	F	32968	BET	40.8	38.6	48	6	52
51	KASIAMMAL	52	M	92616	VAC	45.6	43.7	82	5	28
52	SOKKAN	38	M	34218	VAC	35.4	35.1	84	4	24
53	PUSHPARAJ	50	M	35962	VAC	44.6	43.3	81	3	27
54	RADHAMANI	60	F	101282	VAC	46.7	44.2	86	2	32
55	ARJUNAN	58	M	43211	VAC	42.3	42.1	92	4	31
56	KARUPPASAMY	65	M	44866	VAC	37.1	36.4	94	3	26
57	DHANALAKSHMI	62	F	45962	VAC	39.4	38.8	93	6	22
58	KAMALAKANNAN	55	M	50234	VAC	40.6	39.9	82	4	25
59	PANDIYARAJ	52	F	51432	VAC	35.2	34.6	66	3	36
60	MUTHUMANIKKAM	52	F	102364	VAC	39.7	38.1	76	5	35
61	JOSEPHRAJ	62	M	104928	VAC	42	41.3	89	7	22
62	RATHINAM	64	M	104628	VAC	40.5	39	85	5	28
63	SUSAIRAJ	38	M	76112	VAC	38.1	38	83	4	24
64	CHELLAPANDI	57	M	81132	VAC	42.7	41.8	82	3	25
65	MUTHANDI	52	M	86142	VAC	37.4	36.5	96	4	26
66	SAHUL HAMEED	42	M	78143	VAC	38.4	37.2	99	6	30
67	PONNUTHAI	60	M	98116	VAC	36.7	35.4	100	2	32
68	MUTHALAGU	52	M	86926	VAC	40.6	39.3	92	3	24
69	NALLAN	36	M	86811	VAC	42.5	41.1	87	4	26
70	VADAMALAIYAN	69	M	75282	VAC	43.1	42.8	85	5	20
71	ANANDHAN	40	M	43291	VAC	42.7	41.9	92	4	21
72	KAMALDEVI	32	M	32914	VAC	40.1	39.5	94	2	32
73	RAMAYEE	40	F	56814	VAC	39.4	38.6	81	2	26
74	PALANI	42	F	104328	VAC	38.1	37.8	72	4	27
75	MUTHUSAMY	62	M	45627	VAC	36.8	36.1	90	3	32
76	PERIYATHAMBI	75	M	54328	VAC	43.7	42.1	92	1	28
77	LAKSHMI	60	M	98136	VAC	42.5	41.8	80	2	24
78	SUNDARAM	72	F	99111	VAC	44.6	44.7	86	3	35
79	SELVI	79	M	49132	VAC	39.7	39.5	82	4	21

80	BAKKIYAM	54	F	50162	VAC	37.4	36.8	84	3	22
81	MUTHUSELVI	35	F	55112	VAC	35.2	34.8	92	2	30
82	ALI MOHAMMED	56	M	43149	VAC	43.5	41.9	88	3	26
83	KANDHASAMY	55	M	101862	VAC	41.1	39.6	94	5	24
84	CHITHIRAISELVI	59	F	105282	VAC	39.4	37.6	96	4	28
85	RAMESH	60	M	78963	VAC	41.6	40.2	89	3	26
86	THIYAGARAJAN	53	M	73928	VAC	43.1	42.6	86	2	24
87	SIVAMALAI	36	M	74768	VAC	39.7	39.2	84	3	20
88	MEENAKSHI	42	F	92862	VAC	40.1	39.9	92	2	24
89	GANESAN	58	M	95292	VAC	42.6	41.9	96	3	30
90	MINAR	42	M	102136	VAC	43.6	42.7	100	2	26
91	VELAYUDHAM	44	M	103786	VAC	43.2	42.2	88	2	22
92	SANDHANAM	80	M	109293	VAC	36.5	36.1	90	4	24
93	MUTHUVEERAN	72	M	113962	VAC	37.4	36.6	88	3	26
94	SARAVANAPANDI	66	M	111743	VAC	38.5	38	86	3	28
95	RAMACHANDRAN	55	M	114222	VAC	41.7	40.6	84	2	32
96	VEERAYEE	57	F	111628	VAC	42.8	42.1	93	4	35
97	AROCKIYASAMY	43	M	108323	VAC	36.7	36.1	90	2	30
98	ESWARI	48	F	106628	VAC	43.2	42.6	88	3	26
99	RAVISANKAR	56	M	98999	VAC	40.2	39.8	86	4	27
100	MUNIYAMMAL	72	F	99914	VAC	37.2	36.9	84	3	32

ABBREVATIONS

VAC	:	vacuum assisted closure.
D-O-A	:	date of admission
D-O-D	:	date of discharge
DM	:	Diabetes mellitus
BET	:	Betadine
SSG	:	Split Skin Grafting

MADURAI MEDICAL COLLEGE
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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Saktheeswaran
Course	:	PG in MS., General Surgery
Period of Study	:	2016-2019
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	A comparative study of efficacy of modified vaccum assisted closure using gloves verses Betadine dressing in wound healing
Ethical Committee as on	:	17.07.2018

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
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